U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 01 September 2004

Case No.: 2003-BLA-5472

In the Matter of

BONNIE J. AKERS, o/b/o and Survivor of
ROBERT M. AKERS,
Claimant,

v.

OMAR MINING COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Leonard Stayton, Esq.
For the Claimant

Ashley M. Harman, Esq.
For the Employer

Before: Michael P. Lesniak
Administrative Law Judge

DECISION AND ORDER – DENYING BENEFITS IN MINER'S AND SURVIVOR'S CLAIMS

This case arises from a claim for benefits under the Black Lung Benefits Act, Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 *et seq.* (the Act), and applicable federal regulations, mainly 20 C.F.R. Parts 410, 718 and 725 (Regulations).

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was

caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.[1]

I conducted a formal hearing in Charleston, West Virginia on August 27, 2003, at which all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations.[2] I held the record open for the submission of additional medical evidence.[3] The parties filed a "Joint Stipulation of Objective Evidence" on March 12, 2004. Employer filed a closing brief on April 5, 2004; Claimant filed a closing brief on April 9, 2004.

ISSUES

The contested issues are:

1. Whether the miner has established a material change of condition pursuant to §725.309;
2. The length of the miner's coal mine employment;
3. The number of dependents for purposes of augmentation of benefits in each claim;
4. Whether the miner had pneumoconiosis;
5. Whether the pneumoconiosis arose out of the miner's coal mine employment;
6. Whether the miner was totally disabled;
7. Whether the total disability was due to pneumoconiosis;
8. Whether the miner's death was due to pneumoconiosis.

TR 11.

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[1] Herein, DX = Director's exhibit, EX = Employer's exhibit, CX = Claimant's exhibit, LM = exhibit number within the living miner's claim, TR = transcript of the hearing, BCR = Board-certified radiologist, and B = B reader.

[2] At the hearing, Director's exhibits 1–31, Claimant's exhibits 1 and 2, and Employer's exhibits 1–12 were admitted into evidence without objection. TR 4, 6, and 8.

[3] Post-hearing, Claimant submitted the medical report of Dr. Green on December 19, 2003; the report is marked CX 3 and is hereby admitted into evidence. Employer submitted the 8/25/03 deposition transcript of Dr. Zaldivar on September 12, 2003; the transcript is marked EX 13 and is hereby admitted into evidence. Employer submitted the 9/17/03 deposition transcript of Dr. Naeye on October 1, 2003; the transcript is marked EX 14 and is hereby admitted into evidence. Employer submitted the 9/23/03 deposition transcript of Dr. Castle on November 5, 2003; the transcript is marked EX 15 and is hereby admitted into evidence. Post-hearing, Employer filed a motion to take the additional deposition of Dr. Green. Claimant had no objection to Employer's motion. On January 20, 2004, I issued an Order Allowing Deposition and Extending Filing Schedule. On April 5, 2004, Employer filed the 3/16/04 deposition transcript of Dr. Green; the transcript is marked EX 16 and is hereby admitted into evidence.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History and Factual Background [4]

The miner, Robert M. Akers, filed his first claim for black lung benefits on June 27, 1973. DX 1 (LM 44-1). On February 15, 1980, the claims examiner denied Claimant's claim because he failed to prove any/all elements of entitlement. DX 1 (LM 44-14). No further action was taken on this claim and it was subsequently closed.

The miner filed his second (duplicate) claim for benefits on December 3, 1984. DX 1 (LM 43-1). On April 22, 1985, the claims examiner denied the claim. DX 1 (LM 43-10). No further action was taken on this claim and it was subsequently closed.

The miner filed his third (duplicate) claim for benefits on June 11, 1987. DX 1 (LM 42-1). On December 2, 1987, the claims examiner denied Claimant's claim because Claimant failed to establish any/all elements of entitlement. DX 1 (LM 42-14). The miner disagreed and requested a formal hearing. DX 1 (LM 42-15). A hearing was held on September 25, 1990 in Madison, West Virginia before Administrative Law Judge (ALJ) Charles P. Rippey. DX 1 (LM 42-27). On November 19, 1990, ALJ Rippey issued a Decision and Order — Denying Benefits, finding that there was no medical evidence of pneumoconiosis and no evidence of a pulmonary disability. DX 1 (LM 42-28).

The miner subsequently appealed the decision to the Benefits Review Board ("BRB" or "Board"). On July 13, 1993, the Board issued an Order indicating the miner had submitted additional medical evidence by letter dated 8/6/91 and that the Board construed that additional evidence as a request for modification. The Board then remanded the case to the district director for further proceedings. DX 1 (LM 42-34).

On October 29, 1993, the district director issued a Proposed Decision and Order Denying Request for Modification. DX 1 (LM 42-37). The miner disagreed and requested a formal hearing. The claim was transferred to the Office of Administrative Law Judges (OALJ) on March 3, 1994. DX 1 (LM 42-39).

ALJ Edith Barnett held a hearing on November 1, 1994 in Madison, West Virginia. DX 1 (LM 42-56). On June 13, 1995, ALJ Barnett issued a Decision and Order — Denying Request for Modification. In her decision, ALJ Barnett found no evidence of pneumoconiosis and found that the miner did not have a totally disabling respiratory impairment. DX 1 (LM 42-59). Claimant disagreed and appealed to the BRB. On August 12, 1996, the Board issued a Decision and Order affirming ALJ Barnett's denial of benefits. DX 1 (LM 42-66).

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[4] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718, (*i.e.*, March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal mine dust occurred in West Virginia, this claim arises within the territorial jurisdiction of the United States Court of Appeals for the Fourth Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

The miner filed his fourth (duplicate) and current claim for benefits on March 13, 1998. DX 1 (LM 1). On August 18, 1998, the claims examiner denied the miner's claim for benefits. DX 1 (LM 15). On October 30, 1998, after the submission of additional medical evidence, the claims examiner found that the miner had established 12.8 years of coal mine employment and noted that the initial findings of no entitlement remained unchanged. DX 1 (LM 21). On February 5, 1999, the claims examiner issued another denial of benefits. DX 1 (LM 25). The miner disagreed and requested a formal hearing. DX 1 (LM 26). The claims examiner issued a Memorandum of Conference and Stipulation of Uncontested and Contested Issues on November 1, 1999. DX 1 (LM 37). The miner requested a formal hearing. DX 1 (LM 41).

The miner died on August 13, 2000. DX 13. On October 19, 2000, ALJ Gerald Tierney held a hearing was Madison, West Virginia. After a brief discussion with the parties, ALJ Tierney cancelled the hearing to allow additional time for the parties to review the autopsy evidence.

On April 3, 2001, Claimant Bonnie Akers filed her application for survivor's benefits. DX 3. On April 16, 2001, on Order of Remand was issued by ALJ Daniel L. Leland to allow the widow's claim to be consolidated with the living miner's claim. On November 5, 2002, the district director issued a Decision and Order Denying Benefits in the survivor claim. It was noted that the presence of pneumoconiosis had been established but that death was not due to pneumoconiosis. DX 24. Claimant disagreed and requested a formal hearing. DX 25. The case was transferred to the OALJ on February 21, 2003.

I held a hearing on August 27, 2003 in Madison, West Virginia. Claimant testified that she was married to the miner at the time of his death and has not remarried. TR 12. She noted that their daughter was under the age of twenty-one for a portion of time after the miner filed for benefits in 1998. TR 12. It was noted that the Department of Labor (DOL) had found 18.2 years of coal mine employment but that Employer would only agree to six years. TR 13. Claimant stated that she met the miner in 1952. Claimant reviewed the social security earnings records with counsel. TR 13–18. She noted that the miner worked for six years for Omar Mine from 1978 to 1984. TR 18. The miner's last employer was Zola Mining in 1986, where he only worked for a few months. TR 18. Claimant stated that the United Mine Workers Association credited the miner with eighteen years of coal mine employment. TR 18. She added that all of the miner's coal mine work was underground. TR 19. Claimant stated the miner ran a loader in the mines and was a foreman for Omar Mining. He also ran a buggy to haul the coal. TR 19. She added that all of the miner's work was heavy work. She did not know if the miner wore a respirator but noted that at night when he came home he would have a ring around his mouth. TR 19. Claimant stated that the miner stopped working for Omar Mines in 1984 because he could not breathe. TR 20. The miner had a stroke in 1998 but had breathing problems for years before then. TR 20. Claimant stated that the miner had problems coughing and spitting up and had to sleep on three pillows. Claimant added the miner smoked a half-pack of cigarettes per day until 1984, when he quit smoking. TR 21. She noted the miner was on breathing pills until the time of his death. TR 21–22. The miner also had high blood pressure and a mild heart attack from blood clots. TR 22. Claimant testified that the miner was hospitalized many times over the years at Logan General Hospital for bronchitis and chest pains. TR 23. Claimant stated that her adopted daughter, Bobbi Jo, was born on April 10, 1981 and that she graduated from high school

and went to community college for two years. Bobbi Jo stopped going to college in May of 2002. TR 24. Bobbi Jo would have been twenty-one years old at that time. TR 25. Claimant stated that she and her husband adopted Bobbi Jo on January 26, 1995. TR 25–26.

Duplicate (Living Miner's) Claim

In *Lisa Lee Mines v. Director, OWCP*, 57 F.3d 402 (1995), *aff'd*, 86 F.3d 1358 (4th Cir. 1996), the Court of Appeals for the Fourth Circuit stated that "[t]he purpose of § 725.309(d) is not to allow a claimant to revisit an earlier denial of benefits, but rather only to show that his condition has materially changed since the earlier denial." *Id.* at 406. The court concluded that it would apply the standard set forth by the Court of Appeals for the Sixth Circuit in *Sharondale Corp. v. Ross*, 42 F.3d 993 (6th Cir. 1994) for establishing a "material change in conditions." The *Sharondale* standard requires that the judge consider all of the new evidence to determine whether the miner has proven at least one of the elements previously adjudicated against him.

As will be discussed in more detail *infra*, all of the physicians who submitted the most recent medical reports agreed that the miner had pneumoconiosis. Employer, in its closing brief, does not dispute this issue. *See* Employer's closing brief. Because the miner has established an element of entitlement previously adjudicated against him, he has thus established a material change in conditions pursuant to § 725.309. Accordingly, I will conduct a *de novo* record review to determine whether the miner is entitled to benefits.

Medical Evidence

Pulmonary Function Studies[5]

Exhibit	Date	Age	Height	FEV 1	MVV	FVC	Qualify?
DX 1 (LM 44-7)	4/23/79	48	68"	3.40	117		No
DX 1 (LM 33)	5/7/84	54	67"	3.51	124	4.89	No
DX 1 (LM 43-4)	12/26/84	54	69"	3.58	136.77	4.49	No
DX 1 (LM 42-9)	6/24/87	56	67.5"	2.23	130	3.56	No
DX 1 (LM 42-36)	10/12/93	62	67"	1.96 1.94*	78 71.9*	3.36 3.34*	No No
DX 1 (LM 30)	9/14/94	63	67"	2.87	108	3.83	No
DX 1 (LM 9)	5/1/98	67	68"	2.43 2.68*	65.5 78.2*	3.26 3.36*	No No

[5] Due to the discrepancies in height, qualification of the vent studies will be based on the average height of 67.64 inches. An asterisk (*) indicates a post-bronchodilator value.

Arterial Blood Gas Studies[6]

Exhibit	Date	PO2	PCO2	Qualify?
DX 1 (LM 44-9)	4/23/79	89	35	No
DX 1 (LM 43-6)	12/26/84	78 80*	45 41*	No No
DX 1 (LM 42-11)	6/24/87	82.2	41.7	No
DX 1 (LM 43-32)	5/11/90	102.6	20.6	No
DX 1 (LM 43-32)	4/15/91	80	29.6	No
DX 1 (LM 42-36)	10/12/93	78.1	42.8	No
DX 1 (LM 30)	9/14/94	89	38	No
DX 1 (LM 11)	5/1/98	76	29.5	No
DX 1 (LM 17)	6/18/98	74.9	34.0	No

Medical Reports

Dr. Eduardo Velasco

The medical report of Dr. Velasco is dated April 23, 1979 and appears at DX 1 (LM 44-8). He noted complaints of sputum production and shortness of breath while walking at level and while climbing hills. After performing a physical examination, Dr. Velasco noted no clinical diagnosis related to the cardiopulmonary system.

Drs. K.W. Rectenwald and J.L. Leef

The medical report of Drs. Rectenwald and Leef is dated May 7, 1984 and appears at DX 1 (LM 33). They noted that the miner worked twenty-eight years in the coal mines and had a fifty-pack-year history of smoking. Physical examination revealed normal breath sounds. After reviewing pulmonary function studies and a chest x-ray, the doctors concluded that there was no evidence of occupational pneumoconiosis but that there was a mild pulmonary functional impairment, probably due to chronic cigarette smoking.

[6] An asterisk (*) indicates a post-exercise result.

Dr. Pathom Thavaradhara

The medical report of Dr. Thavaradhara, dated December 26, 1984, appears at DX 1 (LM 43-5). The physician conducted his examination of the miner at the request of the DOL. He noted a medical history of frequent colds, attacks of wheezing, and arthritis. He noted a smoking history of twenty-eight years, ending in 1984. The miner's chief complaints were cough with sputum production, wheezing, dyspnea, chest pain, two-pillow orthopnea, and paroxysmal nocturnal dyspnea. Physical examination showed no signs of congestive heart failure or cor pulmonale. Dr. Thavaradhara concluded that the miner's cardiopulomonary system was normal.

Dr. Armando Acosta/Holden Hospital

The medical report of Dr. Acosta is dated June 24, 1987 and appears at DX 1 (LM 42-10). Dr. Acosta examined the miner at the request of the DOL. He reviewed the miner's occupational history and noted that the miner's last coal mine job with Omar Mining was as a section boss. He noted a history of frequent colds, pneumonia, attacks of wheezing, arthritis, prostate cancer, and heart disease. He noted that the miner had a pulmonary embolism and myocardial infarction in March of 1986. The smoking history was noted as half a pack per day for twenty-five years. The miner's chief complaints were cough with sputum production, wheezing, dyspnea, chest pain, orthopnea, paroxysmal nocturnal dyspnea, and ankle edema. Physical examination of the lungs revealed an increased AP diameter, poor air volume exchange, and mild wheezing with no rales or rhonchi. Dr. Acosta concluded that the miner had mild occupational pneumoconiosis due to coal mine dust exposure.

Drs. A.K. Pfister and J.L. Leef

The medical report of Drs. Pfister and Leef is dated March 10, 1988 and appears at DX 1 (LM 33). It was noted the miner worked thirty years, all inside the mines. The miner's past medical history was positive for pleurisy in 1981, pneumonia in 1982, pulmonary embolism in 1986, elevated blood pressure and mild heart attack in 1984, radical prostatectomy in 1986 for cancer of the prostate, and arthritis in neck and hands. A smoking history was noted as one pack per day for thirty-five years, ending in 1985. Physical examination revealed unimpaired breath sounds. The doctors concluded there was no evidence of occupational pneumoconiosis.

Dr. George L. Zaldivar

The medical report of Dr. Zaldivar is dated September 16, 1994 and appears at DX 1 (LM 30, LM 42-49). Dr. Zaldivar is Board-certified in internal medicine, pulmonary disease, and sleep disorders and is a B-reader of chest x-rays. He reviewed and summarized various medical reports and records. Dr. Zaldivar examined the miner on September 14, 1994. The miner's chief complaint was chest pain and shortness of breath. He also complained of wheezing, ankle edema, two-pillow orthopnea, and cough productive of sputum. A smoking history of one pack of cigarettes per day from the age of seventeen until 1985 was also noted. Dr. Zaldivar reviewed the miner's occupational history and noted twenty-eight years of coal mine employment, ending in 1986. The miner's last job in the mines was as section foreman. The miner stated that the hardest part of the job was changing buggy tires that weighed two hundred pounds. Physical

examination revealed lungs clear to auscultation without wheezes, crackles, or rales. Dr. Zaldivar's impression after the examination was severe hypertension with a history of chest pain on exertion, compatible with angina. Dr. Zaldivar reviewed the objective diagnostic testing and noted no radiographic evidence of pneumoconiosis, normal spirometry, normal arterial blood gases at rest, and normal ECG at rest. Dr. Zaldivar opined that the miner's shortness of breath was due to pressure in the chest wall and that the miner had all of the qualifications for a clinical diagnosis of angina. In conclusion, based on the normal breathing test, normal blood gases, no radiographic evidence of pneumoconiosis, and normal physical examination, Dr. Zaldivar opined that the miner did not have any pulmonary impairment. Based on the past history of heart attack, history of pulmonary embolism, and the clinical diagnosis of angina, Dr. Zaldivar further stated that the miner's sense of shortness of breath and pressure in the chest with exercise was entirely due to cardiac disease caused by coronary artery disease (CAD). Dr. Zaldivar concluded the miner did not have coal workers' pneumoconiosis (CWP) and, from a pulmonary standpoint, was capable of performing arduous manual labor.

The deposition of Dr. Zaldivar was taken on October 17, 1994 and appears at DX 1 (LM 30, LM 42-55). He noted that the vent studies from 1987 were not acceptable because there was a lot of hesitation during the exhalation. He also invalidated the vent studies from 1979 and 1993 because of poor effort on behalf of the miner. The remainder of Dr. Zaldivar's testimony was basically a reiteration of his medical report.

The second medical report of Dr. Zaldivar is dated September 27, 1999 and appears at DX 1 (LM 34). He examined the miner on September 15, 1999 at the request of Employer. He also reviewed and summarized various medical records and reports. At the time of the exam, the miner's wife had to answer all questions because the miner had a stroke in October of 1998 that caused paralysis on the right side of his body and left him unable to speak. The miner's chief complaints were shortness of breath, cerebral vascular accident, and blood clots in his lungs. Other complaints included cough with sputum production, swelling of right leg, and two-pillow orthopnea. It was noted that the miner had stopped smoking thirteen years before and that he had smoked less than a half-pack of cigarettes per day. Dr. Zaldivar noted an occupational history of twenty-eight years of coal mine employment. Physical examination of the lungs revealed crackles with no wheezes. Dr. Zaldivar's impression was CVA with aphasia and paralysis of the right arm and leg, poor peripheral circulation to the legs, inability to cooperate with breathing tests, and past history of smoking. He concluded that there was no evidence of CWP radiographically or by laboratory testing. He added that there no pulmonary or respiratory impairment was present. Dr. Zaldivar agreed that the miner was disabled as a result of the CVA or cardiac disease. He opined that the miner's shortness of breath was the result of severe congestive heart failure as shown by the echocardiograms performed during the past hospitalizations. He added that, even if the miner were found to have CWP, his opinion regarding disability would remain the same.

The deposition of Dr. Zaldivar was taken on October 11, 2000 and appears at EX 12. He noted that, because the miner was so debilitated from his recent stroke during his examination, Dr. Zaldivar could not do any physiologic testing and had to rely on his previous evaluation for physiologic testing in making his determination. The remainder of his testimony was basically a reiteration of his medical report.

Dr. Gregory J. Fino

The medical report of Dr. Fino is dated October 5, 1994 and appears at DX 1 (LM 30; LM 42-53). Dr. Fino, who is Board-certified in internal medicine and pulmonary disease, conducted a medical record review on behalf of Employer. He noted thirty years of coal mine employment. Based on a majority of x-ray readings negative for pneumoconiosis, normal spirometry, normal lung volumes, normal diffusing capacity, and normal arterial blood gases, Dr. Fino opined that the miner did not have pneumoconiosis and, from a functional capacity, had no pulmonary or respiratory impairment. Dr. Fino concluded that there was insufficient objective medical evidence to justify a diagnosis of simple CWP and that in his opinion the miner did not suffer from an occupationally acquired pulmonary condition. He added that no pulmonary impairment was present and that, from a respiratory standpoint, the miner was neither partially nor totally disabled from returning to his last mining job.

The second medical report of Dr. Fino is dated July 3, 2000 and appears at DX 1. He reviewed and summarized additional medical evidence at the request of Employer. He found that the miner did not suffer from CWP based on the majority of chest x-rays being negative for pneumoconiosis, the negative chest x-ray readings, and the normal arterial blood gases. From a functional standpoint, the miner's pulmonary system was normal and retained the capacity to perform the duties of his last coal mine job, even if it required heavy sustained labor. Dr. Fino found that the miner had normal lung function before his stroke and that subsequent to the stroke he was too impaired to do any pulmonary testing. However, based on the lung function studies available, there was no evidence of respiratory impairment. He noted that the miner was disabled as a whole man due to his stroke, significant heart disease, and heart failure. He added that, even if pneumoconiosis were present, there was no evidence of a pulmonary disability.

Dr. Mohammed I. Ranavaya

The medical report of Dr. Ranavaya, dated May 1, 1998, appears at DX 1 (LM 10). He conducted a physical examination of the miner at the request of the DOL. He reviewed the miner's occupational history, noting thirty years of underground coal mine employment with a final position as foreman. Dr. Ranavaya noted a smoking history of one pack of cigarettes per day from age seventeen until 1993. The miner's chief complaints were sputum production, wheezing, dyspnea, cough, chest pain, orthopnea, ankle edema, and paroxysmal nocturnal dyspnea. Physical examination revealed a minimally prolonged respiratory phase with few scattered wheezes. The miner declined an exercise arterial blood gas study due to chest pains and exertional angina. Dr. Ranavaya reviewed the results of a chest x-ray, vent study, arterial blood gas study, and EKG. He concluded that the miner had pneumoconiosis based on thirty years of occupational exposure to coal mine dust and radiological evidence of the disease. He also diagnosed CAD based on history. Dr. Ranavaya concluded that the miner had a mild impairment which, in itself, would not prevent him from performing his usual coal mine work.

Dr. James Castle

The medical report of Dr. Castle is dated August 29, 2000 and appears at EX 10. Dr. Castle is Board-certified in internal medicine and pulmonary disease and is a B-reader of chest x-

rays. Dr. Castle conducted a medical record review at the request of Employer. Dr. Castle reviewed and summarized the medical records in his report. He concluded that based upon a thorough review of all of the data, including medical histories, physical examinations, radiographic reports, pulmonary function tests, arterial blood gases, and other data, the miner did not have pneumoconiosis. Dr. Castle added that at no time did the miner's testing consistently indicate the presence of an interstitial pulmonary process. The miner did not have consistent findings of rales, crackles, or crepitations except when in congestive heart failure. Dr. Castle noted that the physiologic studies showed no evidence of obstruction, restriction, or reduced diffusion capacity. The arterial blood gases were essentially normal. Dr. Castle concluded that the miner was not disabled as a result of any process arising out of his coal mine employment. He added that, from a pulmonary point of view, the miner retained the respiratory capacity to perform his usual coal mine employment. Dr. Castle stated that the miner was totally disabled as the result of his CVA and very likely because of an ischemic cardiomyopathy as well. He added that even if the miner had radiographic evidence of CWP, his opinion regarding the lack of pulmonary impairment would remain unchanged.

Dr. Lawrence Repsher

The medical report of Dr. Repsher is dated September 6, 2000 and appears at EX 11. Dr. Repsher is Board-certified in internal medicine, pulmonary disease, and critical care medicine, and is a B-reader of chest x-rays. Dr. Repsher conducted a medical record review at the request of Employer. He reviewed and summarized the medical information he was provided by Employer. His impression was: (1) no evidence of CWP, (2) no evidence of pulmonary disease, despite a long and heavy cigarette smoking history, (3) hypertension, (4) hyperlipidemia, (5) CAD, complicated by old anterior MI, severe ischemic cardiomyopathy, cardiomegaly, and recurrent congestive heart failure, (6) cerebral vascular accident, (7) degenerative joint disease, (8) status post TURP for carcinoma of the prostate, and (9) probable true posterior acute MI. He opined that the miner never suffered from any pulmonary impairment caused by or aggravated by his coal mine employment. He based his conclusion on the lack of evidence of CWP by x-ray, pulmonary function study, or arterial blood gas, as well as the miner's symptoms of dyspnea on exertion that were more adequately explained by severe ischemic cardiomyopathy, recurrent congestive heart failure, and angina pectoris. Dr. Repsher opined that the miner was totally disabled from performing his last coal mine employment but that this disability was due solely to his massive left CVA and CAD, complicated by severe ischemic cardiomyopathy and recurrent congestive heart failure. The miner's total disability was in no way related to CWP specifically or lung disease generally. Dr. Repsher concluded that even if the miner had microscopic evidence of pneumoconiosis, his opinion regarding the lack of pulmonary impairment would remain unchanged.

Miscellaneous Medical Records

The miner was admitted to Logan General Hospital on June 18, 1998 for treatment of acute congestive heart failure and dyspnea. He was subsequently discharged on June 22, 1998 with acute congestive heart failure, severe hypertension, atherosclerotic heart disease, and LVH. DX 1 (LM 17).

The miner was admitted to HealthSouth on November 9, 1998 and discharged on December 16, 1998 for impaired mobility secondary to left CVA with right hemiparesis. It was noted that the miner's ejection fraction was decreased to 25% as was seen in the EKG.

Death Certificate

The death certificate is dated August 13, 2000 and is certified by Dr. Fernandez of Logan General Hospital. The date of death was listed as August 13, 2000. The cause of death was acute cardiopulmonary arrest and massive acute myocardial infarction. DX 13.

Autopsy

An autopsy report dated September 29, 2000 appears at DX 14. An autopsy was completed on September 28, 2000 by Dr. Carlos Delara. The miner had been brought to the emergency room of Logan General Hospital on August 14, 2000, unresponsive with pupils dilated and fixed. The family requested an autopsy for black lung determination only. In a brief report, the final anatomical diagnosis was noted as (1) acute myocardial infarction, due to arteriosclerotic coronary heart disease and congestive heart failure (clinical), and (2) CWP, moderate. The report notes that the miner had moderate CWP, which was the main reason for his pulmonary problems. Dr. Delara added that the pneumoconiosis was a contributing factor to the immediate cause of death, which was presumably the miner's heart.

Post-Mortem Pathology Reports

Dr. Richard L. Naeye

The medical report of Dr. Naeye is dated February 5, 2001 and appears at EX 1. Dr. Naeye is Board-certified in anatomical and clinical pathology. EX 6. He reviewed various medical records but did not receive a copy of the death certificate or autopsy report. He noted an occupational history of thirty-two years of underground coal mine employment and a smoking history of one pack of cigarettes per day from 1948 to 1984 or 1987, for a total of thirty-six to thirty-nine pack years. He noted that the miner never had significant abnormalities in either lung function studies or arterial blood gas analyses. He added that the many chest x-rays over the years were almost all negative for pneumoconiosis. Dr. Naeye also made note of the miner's history of hypertension, CAD including angina, cardiomegaly with congestive heart failure, pleural effusion, pulmonary edema, and stroke in 1998. He reviewed ten slides taken from the autopsy containing lung tissue. He noted that there was a small to moderate amount of black pigment in the lung tissue. He identified the presence of anthracotic micronodules and some anthracotic macules. He stated that the micronodules and a few of the macules had thin rims of focal emphysema around them, constituting about 1% of the total emphysema in the lung tissues. All of the other emphysema present was mild in severity and centrilobular in type. There was microscopic evidence of chronic bronchitis and widespread atelectasis.

Dr. Naeye concluded that mild simple CWP was present in the form of a small number of anthracotic micronodules and macules with accompanying fibrosis. He added that the clinical insignificance of these findings was confirmed by the absence of CWP on chest x-rays, as well as the normal PFTs and arterial blood gases as late as 1998—twelve years after the miner left the coal mines. He opined that the miner's CWP did not cause any measurable impairment in lung function, any disability, or contribute in any way to the miner's death. He stated that the miner was disabled by cardiac failure and, in 1998, by a stroke. Dr. Naeye added that, while the miner's many years of cigarette smoking did not cause any impairment in lung function, they likely played a role in the miner's death by damaging the microcirculation of the heart, which contributed to his progressive cardiac dysfunction and failure. The physician stated that microscopic examination of the lung tissue showed plugging of small airways by mucous, causing widespread atelectasis. He noted that this latter event was caused at least in part by chronic bronchitis due to cigarette smoking. He concluded that smoking rather than industrial bronchitis was the cause of the miner's terminal mucous plugging of small airways.

Prior to his deposition, which was taken September 17, 2003 and appears at EX 14, Dr. Naeye reviewed the autopsy report and death certificate. He agreed that the PO2 of 76 when the miner was sixty-seven years old was within the normal range. He noted that the PO2 of 74.9 in 1998 was borderline between completely normal and maybe abnormal. The medical evidence showed that the miner had extensive damage in the coronary arteries and in the microcirculation of the heart. He added that the fluid in the lungs was secondary to heart failure. He said that the microscopic findings met the minimum requirements to diagnose CWP but that the damage was not severe enough to have caused any abnormalities in lung function or to play any role in causing death. Dr. Naeye disagreed with the opinion of Dr. Green, noting that the miner's PO2 values were not very abnormal and that, if the miner had not had heart disease, he would not have been affected by those levels whatsoever. He concluded that the miner died as a consequence of his cigarette smoking and the tremendous damage this did in the microcirculation of his heart. He stated that he attributed the mucous plugging of the small airways to smoking rather than industrial bronchitis because, in studies, people with bronchitis who have never smoked have a recession of the bronchitis over a period of time. He added that, in his experience, once a miner has been out of the mines for four to five years, he does not have significant microscopic evidence of chronic bronchitis.

Dr. P. Raphael Caffrey

The medical report of Dr. Caffrey is dated February 20 ,2001 and appears at DX 23. Dr. Caffrey is Board-certified in anatomical and clinical pathology. DX 23. He reviewed various medical records, including the autopsy report and ten autopsy slides containing lung tissue. His examination of the tissue revealed a mild amount of anthracotic pigment. On the right side of the lung, he noted a few lesions of simple CWP with an occasional micronodule, acute passive congestion, focal atelectasis, and a mild degree of centrilobular emphysema. The left side of the lung showed minimal anthracotic pigment with few lesions of simple CWP, focal edema fluid, and acute passive congestion. Dr. Caffrey noted an occupational history of thirty-two years of coal mine employment and a forty-six-pack-year history of smoking, ending in 1993. He reviewed the miner's medical history and objective diagnostic testing. Dr. Caffrey opined that the miner had a mild degree of simple CWP and disagreed with the autopsy prosector who

diagnosed moderate CWP. Dr. Caffrey based this opinion on the gross examination, which noted the presence of some anthracotic pigment but described no nodules. He added that, at most, he identified four lesions of simple CWP on any one slide, which would have occupied no more than 10% of lung tissue and that, at on least one slide, he found no lesions of simple CWP. Dr. Caffrey stated that most of the x-ray interpretations were negative, which fit closely with his opinion of mild simple CWP. He also opined that the miner had a mild degree of centrilobular emphysema due to the miner's forty-plus years of smoking cigarettes. He added that the weight of the lungs on gross examination correlated well with someone in congestive heart failure. Dr. Caffrey concluded the miner had mild simple CWP but that the CWP did not cause him pulmonary or respiratory impairment prior to death and did not cause, or contribute to, his hypertension, cardiac disease, or CVA. It was his opinion that the miner's death was due to atherosclerosis that affected the miner's coronary arteries, heart, and brain, which resulted in acute congestive heart failure, severe hypertension, and a large ischemic infarct within his brain.

Dr. Erika C. Crouch

The medical report of Dr. Crouch is dated March 20, 2001 and appears at DX 22. Dr. Crouch is Board-certified in anatomic pathology. She reviewed the autopsy slides, autopsy report, death certificate, and miscellaneous medical records of the miner as well as the reports of Drs. Caffrey, Naeye, and Fino. She noted that the autopsy slides showed no acute disease with only mild centriacinar emphysema. She identified small numbers of small coal dust macules characterized by coal dust containing macrophages but generally mild associated fibrosis. She added that most of the parenchyma appeared entirely unremarkable. Dr. Crouch diagnosed mild centriacinar emphysema and mild simple CWP. She stated that there was histological evidence of mild simple CWP with small numbers of small coal-dust-related lesions. She opined that coal dust exposure could not have caused any degree of functional impairment and could not have contributed to any known disability. She noted that the miner had suffered a stroke and had documented cardiovascular disease, indicating that any impairment was most likely neurologic and/or cardiac in origin, and secondary to atherosclerotic cardiovascular disease. Dr. Crouch stated that coal dust exposure does not contribute to heart disease in the absence of severe pulmonary impairment and does not increase the risk of stroke. Therefore, she concluded that occupational coal mine dust exposure could not have caused or hastened the miner's death.

The supplemental report of Dr. Crouch, dated July 31, 2003 and appearing at EX 7, reviews the 7/9/03 medical report of Dr. Green. Dr. Crouch stated that her opinion had not changed in any way. She noted that they agreed that the miner had CWP but differed as to the severity of the disease. Dr. Crouch disagreed that CWP contributed to death by causing hypoxemia. She reiterated that the changes were of insufficient severity to have caused a clinically significant degree of functional impairment. She added that any clinical evidence of hypoxemia could reasonably be attributed to the miner's documented cardiac disease.

Dr. Francis H.Y. Green

The medical report of Dr. Green is dated July 9, 2003 and appears at CX 1. Dr. Green, who is Board-certified in anatomic pathology, reviewed the miner's autopsy slides, autopsy report, death certificate, and the medical reports of Drs. Crouch, Caffrey, Zaldivar and Fino. In

addition, Dr. Green reviewed various miscellaneous medical records. Dr. Green stated that microscopic review of the autopsy slides revealed the presence of moderately severe CWP comprising a combination of silicotic and mixed dust nodules with coal dust macules and associated emphysema. He noted that the lesions were scattered throughout the lungs. In addition to pneumoconiosis, there was also evidence of chronic bronchitis characterized by the increase in size of mucous glands with an increased ratio of mucous to serous cells. In addition, the lungs showed evidence of pulmonary edema. Dr. Green diagnosed the miner with moderately severe simple CWP comprising silicotic and coal dust nodules and macules with focal edema, chronic bronchitis and emphysema, and pulmonary edema.

A review of the medical record revealed that the likely cause of death was congestive heart failure secondary to underlying cardiac disease. Dr. Green confirmed that the miner had moderate CWP due to exposure to coal mine dust. The emphysema and chronic bronchitis were due in part from exposure to coal mine dust and in part to the miner's smoking history that ranged from twelve-and-a-half pack years to over forty pack years. Dr. Green opined that the presence of moderately severe CWP combined with dust-induced bronchitis and emphysema contributed to the miner's death by causing or increasing hypoxemia. He noted that the miner's last PO2 value in 1998 was abnormally low. He stated that the pneumoconiosis would have contributed to the low PO2, which would have made the heart more susceptible to the effects of hypoxemia. He added that coronary artery atherosclerosis resulted in poor perfusion and oxygenation of the cardiac muscle; therefore, a low oxygen saturation in the blood perfusing the heart would contribute to the onset of a fatal arrhythmia or acute myocardial infarction.

The supplemental report of Dr. Green is dated December 17, 2003 and appears at CX 3. Dr. Green reviewed the depositions of Drs. Zaldivar, Naeye, and Castle. He disagreed with Dr. Zaldivar and maintained that the arterial blood gases were abnormally low. He noted that he agreed with Dr. Naeye's opinion in many respects: that the miner had CWP, that the miner had low normal or abnormal PO2 values, and that the mechanism of death was related to the effects of lung disease on the heart. Dr. Naeye attributed all of the heart disease to cigarette smoking, while Dr. Green attributed a significant portion to the effects of CWP. Dr. Green stated that Dr. Castle was confusing an abnormal laboratory result with a disease requiring treatment. He added that although the miner's hypoxemia was not low enough to cause him significant respiratory impairment or require him to receive supplemental oxygen, it was sufficient to increase the risk of his developing ischemic heart disease due to the fact that the lower oxygen tension in the miner's blood would reduce the amount of oxygen going to the myocardium. He concluded that lung disease, whether it was due to cigarette smoking or pneumoconiosis, would accelerate ischemic heart disease and the resulting hypoxemia could contribute to impaired oxygenation of vital tissues such as the heart, especially if they have pre-existing CAD.

The deposition of Dr. Green was taken on March 16, 2004 and appears at EX 16. He noted that the degree of CWP present should have appeared on x-rays. He agreed that there was a cardiac-care mechanism of death. He noted that, because the heart was not examined at autopsy, there is an impediment to knowing exactly the mechanism or cause of death. He noted the presence of pulmonary edema in the slides, which is consistent with congestive heart failure. He agreed that there was no reason to connect atelectasis with pneumoconiosis. Dr. Green could not recall how many CWP lesions he found per slide. He noted that smoking and coal mine dust

exposure cause emphysema and chronic bronchitis. He noted that it is not possible to differentiate the etiology of chronic bronchitis and emphysema between smoking and coal dust exposure; therefore, he had to include both. Dr. Green did not have any information about the dust levels the miner was exposed to in the mines. He agreed that his only clinical experience treating patients with pulmonary problems was during his internship in the early 1970's.

Other Medical Reports

Dr. George L. Zaldivar

For his supplemental medical report dated April 7, 2001 and appearing at EX 2, Dr. Zaldivar reviewed additional information including the medical reports of Drs. Fino, Repsher, Naeye, Caffrey, and Crouch as well as the autopsy report and death certificate. He noted that the additional evidence established CWP microscopically but that the miner's breathing tests showed no measurable pulmonary dysfunction. He concluded that (1) there was objective evidence to justify a diagnosis of CWP, (2) there was no pulmonary impairment present prior to the stroke, (3) the miner was totally and permanently disabled from working, although the impairment was the result of the CVA, coronary vascular disease and angina, and (4) the microscopic CWP found at autopsy was incidental in the sense that the CWP did not cause any pulmonary dysfunction in life. He concluded that the CWP did not cause or contribute to the miner's death in any way, and that the miner would have died when and as he did even if he had never worked in the mines because his death was due to cardiovascular disease unrelated to his occupation.

The deposition of Dr. Zaldivar is dated August 25, 2003 and appears at EX 13. He stated that, regardless of the cause of the microscopically detected emphysema, there was no evidence that it caused any pulmonary impairment. He added that the pulmonary edema described by the pathologist was due to the miner's cardiac problems and was unrelated to pneumoconiosis. He opined that the miner suffered a cardiac death either by heart attack or cardiac arrhythmia that caused cardiac standstill. Dr. Zaldivar noted that the records showed chronic pulmonary edema because the miner's heart was working so poorly. He stated that Dr. Green was incorrect in concluding that the miner had hypoxemia. Dr. Zaldivar stated that the miner's PO2 was normal. He added that it was "nonsense" to say that a PO2 in the seventies will result in any kind of cardiac irregularity because of hypoxemia. He acknowledged that a stroke could make pulmonary function decline because of weakness of the respiratory muscles—a neuromuscular problem, not a pulmonary intrinsic problem. He stated that it did not matter whether the CWP was classified as mild or moderate because there was no pulmonary abnormality present. He added that a diagnosis of chronic bronchitis was appropriate in this case but disagreed that it was related to coal dust exposure. He noted that the CWP macules were scarred and were not actively irritating the airways. Dr. Zaldivar opined that CWP cannot cause any pulmonary impairment in the absence of any physiologic abnormality by breathing tests or blood gases.

Dr. James R. Castle

For his supplemental medical report dated April 11, 2001 and appearing at EX 3, Dr. Castle reviewed the medical reports of Drs. Fino, Repsher, Naeye, Caffrey, and Crouch, the

autopsy report, and the death certificate. He admitted that there was pathologic evidence of minimal, simple CWP, adding that three very experienced pathologists indicated it was minimal in nature and could not have contributed to any impairment or disability during life and did not hasten the miner's death in any way. He noted that the minimal CWP did not cause the miner to have an abnormal chest x-ray in the opinion of the vast majority of B-readers. Dr. Castle added that it was important to note the lack of evidence of any respiratory impairment from any cause based on pulmonary function studies and blood gas tests. Dr. Castle opined that the miner had no pulmonary disability due to CWP during his life and that the miner was disabled as a result of cerebrovascular disease, CAD with ischemic cardiomyopathy, and congestive heart failure. Moreover, he opined that the miner's death was not caused by, contributed to, or hastened by the underlying pathologic CWP, as the changes present were so minimal as to have neither caused disability during his lifetime nor hastened death. He concluded that the miner died as a result of acute myocardial infarction in the setting of severe ischemic cardiomyopathy and severe cerebrovascular disease.

During the deposition of Dr. Castle taken on October 19, 2003 and appearing at EX 15, he disagreed with Dr. Green's opinion that the last ABG study showed evidence of hypoxemia. He stated that the value in question was normal for the miner's age and did not indicate any degree of hypoxemia. He concluded that the miner had no significant pulmonary impairment prior to death and that the miner's pneumoconiosis played no role in his death. He stated that, if the pneumoconiosis did not cause pulmonary impairment or any blood gas abnormalities, then he did not see any way that it could have contributed to death.

Dr. Gregory J. Fino

The medical report of Dr. Fino is dated April 11, 2001 and appears at EX 4. Dr. Fino reviewed additional information including the death certificate, the autopsy report, and the medical reports of Drs. Naeye, Caffrey, Crouch, Castle, and Repsher. Dr. Fino stated that there is no increased incidence of CAD or its sequelae in individuals who work in the coal mines or who have CWP. There is no increased incidence of sudden death due to heart disease or sudden cardiac arrest in coal miners or those who have CWP. Finally, he added that there is no increased incidence of cardiac arrthymias in those with chronic hypoxemia due to chronic fibrotic conditions of the lungs, of which CWP is an example. Based on the information he had reviewed, Dr. Fino opined that the miner would have died as and when he did had he never stepped foot in the mines, or had he never contracted CWP. He noted that the CWP found at autopsy was subclinical, meaning that it did not cause a respiratory impairment. He concluded that the simple CWP did not cause, contribute to, or hasten the miner's death.

The supplemental report of Dr. Fino is dated August 5, 2003 and appears at EX 9. Dr. Fino reviewed the 7/9/03 medical report of Dr. Green. He disagreed with Dr. Green's comment that the PO2 value in 1998 was abnormally low. He noted that for a sixty-eight-year-old man, a PO2 of 74.9 was completely normal and that there was no hypoxemia. Dr. Fino did not change any of his original opinions in this matter.

Conclusions of Law

Length of Coal Mine Employment

At the hearing, Employer would only stipulate to the six years the miner worked at Omar Mining Company. TR 11. Claimant is alleging over thirty years of coal mine employment. The DOL prepared a worksheet based on the miner's earnings and number of days worked to calculate the length of coal mine employment. In this case, the DOL could verify 18.02 years of coal mine employment. DX 8.

In its Proposed Decision and Order Denying Benefits, the district director found that Claimant had established eighteen years of coal mine employment. DX 24. As part of her application for benefits, Claimant submitted a detailed work history for the miner which, I find, supports the district director's finding of eighteen years of coal mine employment. DX 5. In addition, Claimant testified at length at the hearing regarding the miner's coal mine employment. TR 13–18. I find Claimant's testimony to be credible and consistent with the miner's social security records. *See also* DX 7. Accordingly, based on the foregoing, I find that Claimant has established eighteen years of qualifying coal mine employment.

Date of Filing

I find that the miner filed his living miner's claim for benefits under the Act on March 13, 1998. TR 11, DX 1 (LM 1) and that Claimant filed her survivor's claim for benefits under the Act on March 31, 2001. TR 11, DX 3.

Responsible Operator

The parties have stipulated and I find that Omar Mining Company is the responsible operator and will provide payment of any benefits awarded to the miner and/or Claimant. TR 11.

Dependents

Living Miner's Claim

Initially, the miner only noted one dependent for purposes of augmentation of benefits: his wife, Bonnie. At the hearing, Employer stipulated that Bonnie was a dependent of the miner. TR 11. However, there was testimony at the hearing by Claimant that she and the miner had adopted a daughter on January 26, 1995. TR 25-26, DX 12. Their daughter, Bobbi Jo, was born on April 10, 1981 and would have been sixteen years old at the time the miner filed his living miner's claim for benefits on March 13, 1998.

Because Bobbi Jo is a qualifying (adopted) child of a beneficiary and was under the age of eighteen, I find that Bobbi Jo is a dependent for purposes of augmentation of benefits. 20

C.F.R. §§ 725.208, 725.209(a)(1), and 725.209(a)(1)(i). In the event of an award in the living miner's claim, the period of entitlement would be limited from March of 1998 (the date of application) until the miner's death on August 13, 2000. At the time of the miner's death, Bobbi Jo would have been nineteen years old. However, there was testimony by Claimant that Bobbi Jo attended community college until May of 2002, which still qualifies her as a dependent under the Act. 20 C.F.R. § 725.209(a)(2)(iii). Accordingly, I find that in the event of an award in the living miner's claim, the miner had two dependents for purposes of augmentation of benefits: his wife, Bonnie, and their daughter, Bobbi Jo.

Survivor's Claim

Likewise, in the survivor's claim, because Bobbi Jo was nineteen years old and attending college at the time of the miner's death, she is considered a dependent of Claimant for purposes of augmentation of benefits. 20 C.F.R. § 725.209(a)(2)(iii). Claimant testified at the hearing that Bobbi Jo attended college until May of 2002, when she was twenty-one years old. Accordingly, I find that in the event of an award in the survivor's claim, Claimant had one dependent for purposes of augmentation of benefits, her daughter, Bobbi Jo, for the period from August 13, 2000 (date of miner's death) until May of 2002 (when Bobbi Jo left college).

Entitlement: Living Miner's Claim

Smoking History

There were differing smoking histories reported throughout the record that ranged from twelve-and-a-half pack years to fifty pack years. However, the majority of physicians noted a range of thirty-five to forty-six pack years, ending around 1986. DX 1 (LM 33, LM 30, LM 10), EX 6, DX 23. Claimant testified at the hearing that the miner smoked half of a pack of cigarettes per day until 1984 but did not indicate when the miner started smoking. TR 21. Therefore, based on the foregoing, I find the miner had a substantial and prolonged smoking history of thirty-five to forty-six pack years, ending around 1986.

Determination of Pneumoconiosis

Thirty U.S.C. § 902(b) and 20 C.F.R. § 718.201 define pneumoconiosis as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment."[7] The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis.[8] 20 C.F.R. § 718.201. The term "arising out of coal

[7] Pneumoconiosis is a progressive and irreversible disease; once present, it does not go away. *Mullins Coal Co. v. Director, OWCP,* 484 U.S. 135, 151 (1987); *Lisa Lee Mines v. Director*, 86 F.3d 1358 (4th Cir. 1996) (*en banc*) at 1364; *LaBelle Processing Co. v. Swarrow*, 72 F.3d 308 (3d Cir. 1995) at 314–315.

[8] Regulatory amendments, effective January 19, 2001, state:

mine employment" is defined as including "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."

As noted previously, the autopsy showed undisputed evidence of pneumoconiosis. All of the pathologists and physicians who reviewed the autopsy report and/or slides agreed that pneumoconiosis was present. Employer did not dispute this issue in its closing brief. Accordingly, I find pneumoconiosis has been established pursuant to § 718.201.

Cause of Pneumoconiosis

Once it is determined that the miner suffers from pneumoconiosis, it must be determined whether the miner's pneumoconiosis arose, at least in part, out of coal mine employment. 20 C.F.R. § 718.203(a). If a miner who is suffering from pneumoconiosis was employed for ten years or more in the coal mines, there shall be a rebuttable presumption that the pneumoconiosis arose out of such employment.

I find that the miner, with eighteen years of coal mine employment, is entitled to the rebuttable presumption at § 718.203. Employer has not set forth any credible evidence to rebut that presumption. Accordingly, the miner has established that his pneumoconiosis arose, at least in part, out of his coal mine employment.

(a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.

(1) "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.

(2) "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

Total Disability

A miner is considered totally disabled if he has a pulmonary or respiratory impairment which, standing alone, prevents him from performing his usual coal mine work or comparable employment. 20 C.F.R. § 718.204(b)(1). Section 718.204 sets out the standards for determining total disability. This section provides that, in the absence of contrary probative evidence, evidence that meets the quality standards of the subsection establishes total disability.

Subsection 718.204(b)(2)(i) provides that total disability may be established by pulmonary function testing. There are seven pulmonary function studies submitted as part of the living miner's claim for benefits. None of the studies produced qualifying values. Therefore, I find that the miner has failed to establish total disability due to § 718.204(b)(2)(i).

Subsection 718.204(b)(2)(ii) provides that qualifying arterial blood gas testing may establish total disability. There are nine arterial blood gas studies in the record. None of the studies were qualifying under the Act. Accordingly, I find that the preponderance of the evidence fails to establish total disability pursuant to § 718.204(b)(2)(ii).

There is no evidence that the miner suffered from cor pulmonale with right-sided congestive heart failure pursuant to § 718.204(b)(2)(iii).

Subsection 718.204(b)(2)(iv) provides that total disability may be established if a physician exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that the miner's respiratory or pulmonary impairment prevents him from engaging in his usual coal mine work or in comparable and gainful employment.

Thirteen physicians have rendered an opinion in this matter relative to this issue. Drs. Velasco, Thavaradhara, Acosta, and Pfister/Leef found either that the miner had normal cardiopulmonary function or offered no opinion regarding the presence of any pulmonary impairment. Drs. Zaldivar, Fino, Ranavaya, Castle, Repsher, Naeye, Caffrey, and Crouch found that the miner had no evidence of any pulmonary impairment.[9] Drs. Rectenwald/Leef found that the miner had a mild pulmonary impairment probably due to chronic cigarette smoking but did not indicate whether the impairment would have prevented him from performing his usual coal mine employment. For this reason, I accord the opinion of Drs. Rectenwald/Leef less weight.

In particular, I accord great weight to the opinions of the highly qualified Drs. Zaldivar, Fino, Ranavaya, Castle, and Repsher on this issue. Each physician is highly skilled with a specialty in the area of pulmonary medicine. I find that their opinions are consistent with the objective diagnostic testing of record, the miner's extensive medical history of severe cardiac disease and stroke, the miner's subjective complaints, occupational history, smoking history, and physical examinations. Moreover, I find that their opinions are supported by the conclusions of

[9] Although Dr. Green provided two comprehensive reports (CX 1, CX3) in this matter, he did not address the issue of whether the miner had a totally disabling pulmonary impairment during his lifetime. In fact, in his supplemental report, Dr. Green acknowledged that the miner's hypoxemia was not low enough to cause significant respiratory impairment or require him to receive supplemental oxygen. CX 3.

three highly qualified pathologists (Drs. Naeye, Caffrey, and Crouch), who also rendered reports in this matter. I also find that there is no contrary medical opinion that supports the assertion that the miner suffered from some pulmonary impairment during his lifetime that would have prevented him from performing his last coal mine employment. Because all of the medical experts agree that the miner did not suffer from any pulmonary impairment that would have prevented him from performing his usual coal mine employment, I find that the miner has failed to establish total disability pursuant to § 718.204(b)(2)(iv) .

In weighing all of the foregoing, I find the miner has failed to establish the existence of a totally disabling respiratory impairment pursuant to § 718.204(b).

Disability Causation

The final issue in the living miner's claim is whether the miner has established disability causation at § 718.204(c)(1).

Pursuant to § 718.204(c)(1), a miner is considered totally disabled due to pneumoconiosis if the disease is a substantially contributing cause of the miner's totally disabling respiratory impairment. Pneumoconiosis is a "substantially contributing cause" of disability if it:

(i) Has a material adverse effect on the miner's respiratory or pulmonary condition; or
(ii) Materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment.

Because I have found that the evidence of record fails to establish, by a preponderance of the evidence, that the miner had a totally disabling respiratory impairment, I accordingly find that the miner has failed to establish that he suffered from a total respiratory disability as a result of having CWP, as required by the Act and Regulations. Again, I credit the opinion of Drs. Zaldivar, Fino, Ranavaya, Castle, and Repsher (*i.e.*, that Claimant suffered from no pulmonary or respiratory impairment due to pneumoconiosis) on the basis of the well-reasoned, thorough nature of their reports.

Conclusion

Because the miner has failed to establish all elements of entitlement, I must conclude that he has not established entitlement to benefits under the Act.

Attorney's Fee

The award of an attorney's fee under the Act is permitted only in cases in which the miner is found to be entitled to benefits. Because benefits are not awarded in this claim, the Act prohibits the charging of any fee to the miner for representation services rendered in pursuit of it.

Entitlement: Survivor's Claim

Death Due to Pneumoconiosis

The sole issue is whether the miner's death was due to pneumoconiosis. Death due to pneumoconiosis may be established under § 728.205(c) by any one of the following criteria:

1. Competent medical evidence establishes that pneumoconiosis was the cause of the miner's death.

2. Evidence that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or that death was caused by complications of pneumoconiosis.

3. Under § 718.304, the miner suffered from a chronic dust disease of the lung and chest X-ray evidence shows one or more large opacities (greater than 1 centimeter), biopsy or autopsy shows massive lesions in the lung, or other evidence (in accord with acceptable medical procedures) shows a condition which could reasonably be expected to yield such large opacities or massive legions.

Section 718.205 (c)(5) provides that pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. 20 C.F.R. § 718.205(c)(5).

There is no credible evidence that pneumoconiosis was the direct cause of the miner's death, therefore Claimant has not proven death due to pneumoconiosis pursuant to § 718.205(c)(1). There is no evidence the miner suffered from complicated pneumoconiosis, therefore Claimant has not established death due to pneumoconiosis pursuant to § 718.205(c)(3).

Eight physicians rendered an opinion on this issue. Drs. Delara and Green opined that CWP was a substantial contributing factor to the miner's death while Drs. Naeye, Caffrey, Crouch, Zaldivar, Castle, and Fino maintained that the miner's CWP played no role in his death due to cardiac disease.

I accord less weight to the opinion of the autopsy prosector, Dr. Delara, on this issue. Dr. Delara noted that the miner had moderate CWP and that the pneumoconiosis was a contributing factor to the immediate cause of death, which was presumably due to the miner's heart. Because Dr. Delara provided no reasoning or rationale for his opinion, I find that his conclusion is not well-reasoned and not well-documented and is thereby accorded less weight.

I accord greater weight to the opinions of Drs. Naeye, Caffrey, and Crouch. All three are highly qualified, Board-certified pathologists. I find their reports to be well-reasoned and well-documented. All agreed that the miner had pathological evidence of simple CWP that was too mild to have caused any abnormalities in lung function. Dr. Caffrey noted that each slide had, at most, four lesions of CWP that would have occupied no more than 10% of lung tissue and that, on at least one slide, he found no lesions of CWP. Dr. Crouch noted that most of the lung parenchyma appeared entirely unremarkable. Dr. Naeye noted that the thin rims of focal emphysema around the micronodules and a few of the macules constituted about 1% of the total emphysema

in the lungs. Drs. Naeye and Caffrey also noted that most of the miner's chest x-rays were read as negative for CWP, which would fit closely their assessment of mild simple CWP.

Conversely, Dr. Green, who is also a highly qualified pathologist, opined that the miner had moderately severe CWP. Likewise, Dr. Delara also noted moderate pneumoconiosis. However, neither Dr. Green nor Dr. Delara adequately articulated the basis for these conclusions. Dr. Green admitted at his deposition that he could not recall how many CWP lesions he found per slide. I also note that the finding of moderately severe pneumoconiosis does not square with the mostly negative x-ray interpretations of record. At his deposition, Dr. Green admitted that the degree of pneumoconiosis that he found to be present should have appeared on x-rays. Accordingly, I find the classification of pneumoconiosis by Drs. Delara and Green as moderate or moderately severe to be unsubstantiated and unconvincing.

I accord greater weight to the opinions of Drs. Zaldivar, Castle, and Fino. All three are Board-certified in internal medicine and pulmonary disease and are experts in the field of pulmonary medicine. Their reports are well-reasoned and well-documented and are supported by the objective diagnostic evidence of record that showed no evidence of an obstructive or restrictive defect, the results of the majority of the reviewing pathologists who noted the presence of only mild simple pneumoconiosis, the miner's medical history of severe cardiac disease and stroke, and the miner's subjective complaints. Moreover, I find that their persuasive opinions outweigh Dr. Green's finding that the miner's mild hypoxemia was sufficient to increase the risk of his developing ischemic heart disease, due to the fact that lower oxygen tension in the miner's blood would reduce the amount of oxygen going to the myocardium. Drs. Zaldivar, Castle, and Fino agreed that Dr. Green was incorrect in concluding the miner had hypoxemia. All three agreed that the results of the ABG study in question were normal for the miner's age and did not indicate any hypoxemia. As noted previously, Drs. Zaldivar, Castle, and Fino have an expertise in treating patients with pulmonary disease. On the other hand, Dr. Green, a pathologist, admitted at his deposition that his only clinical experience treating patients with pulmonary problems was during his internship in the early 1970's. Accordingly, I find the opinions of Drs. Zaldivar, Castle, and Fino to be more credible than the opinion of Dr. Green.

I accord less weight to the opinion of Dr. Green. I find that his report is not well-reasoned or well-documented. As noted previously, Dr. Green based his opinion, at least in part, on his finding of moderately severe CWP. As I discussed above, I find that this conclusion is not supported by the better evidence of record. Moreover, the cornerstone of Dr. Green's opinion was that the miner suffered from mild hypoxemia due to the moderately severe CWP that would have increased his risk for developing ischemic heart disease by ultimately reducing the amount of oxygen going to the myocardium. However, this theory was debunked by the well-reasoned, well-documented opinions of Drs. Zaldivar, Castle, and Fino, who found that the miner did not suffer from any hypoxemia.[10] Based on the foregoing, I find that the opinion of Dr. Green is not well-reasoned or well-documented and is therefore accorded less weight.

[10] Dr. Naeye, a pathologist, stated at his deposition that the arterial blood gas study in question was borderline between completely normal and was possibly abnormal. I find that Dr. Naeye's statement regarding this arterial blood gas study is not credible in light of the persuasive conclusions of three highly qualified pulmonologists who opined to the contrary. Nevertheless, I find Dr. Naeye's opinion to be otherwise well-reasoned.

Based on the above, I find that Claimant has failed to establish that the miner's death was due to pneumoconiosis pursuant to § 728.205(c).

Conclusion

Because Claimant has failed to establish all elements of entitlement, I must conclude that she has not established entitlement to benefits under the Act.

Attorney's Fee

The award of an attorney's fee under the Act is permitted only in cases in which Claimant is found entitled to benefits. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for representation services rendered in pursuit of the claim.

ORDERS

Living Miner's Claim

The claim of BONNIE J. AKERS O/B/O ROBERT M. AKERS for black lung benefits under the Act is hereby DENIED.

Survivor's Claim

The claim of BONNIE J. AKERS, as surviving spouse of ROBERT M. AKERS, for black lung benefits under the Act is hereby DENIED.

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MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days of the date this Decision and Order was filed in the office of the District Director, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.